Figure 1

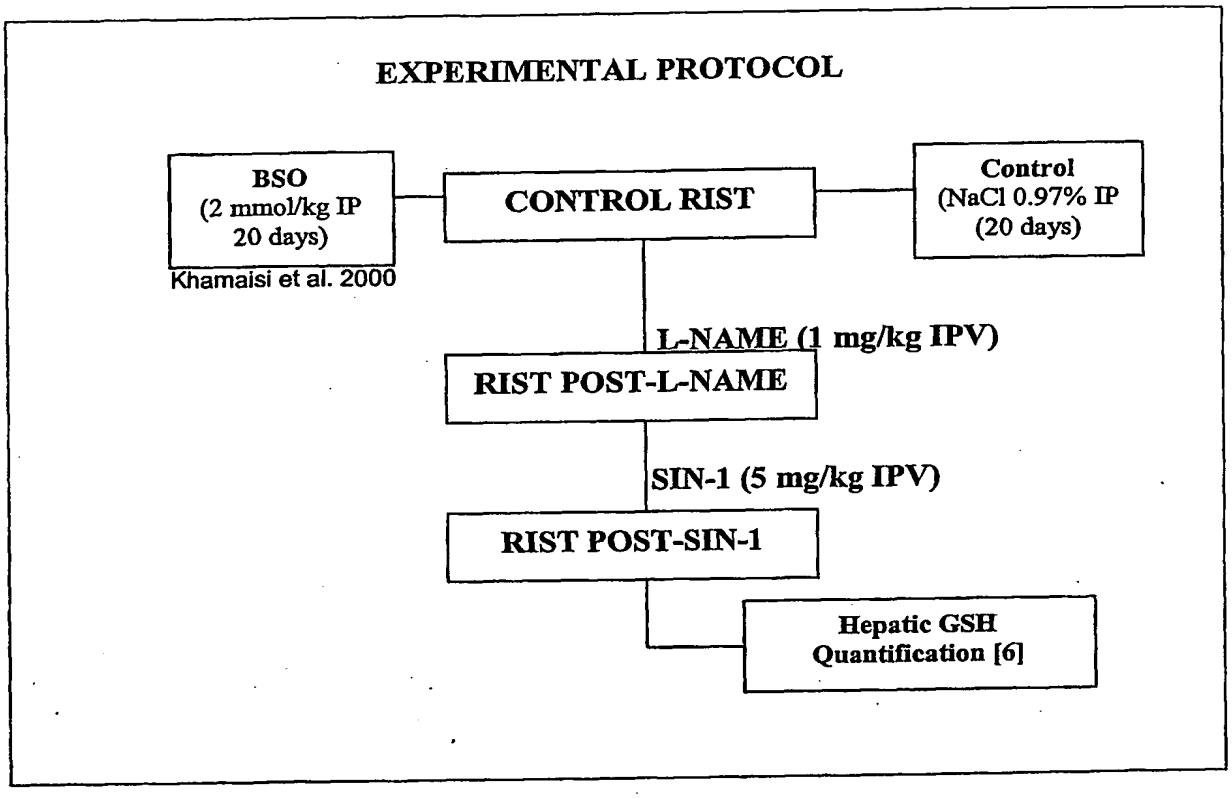
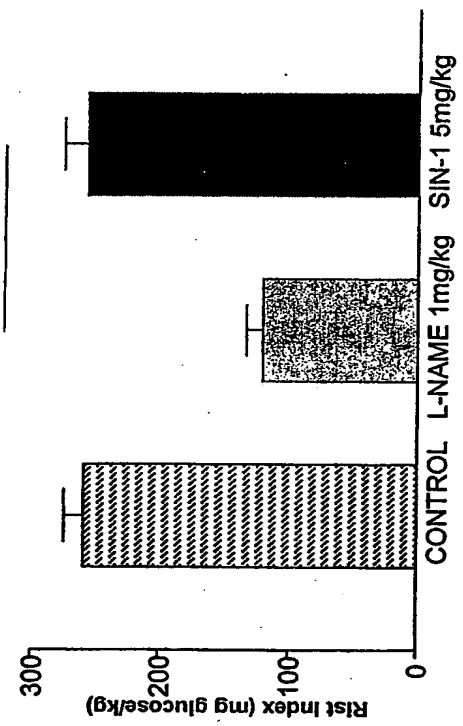

EXPERIMENTAL PROTOCOL
BSO
(2 mmol/kg IP
20 days)
Khamaisi et al. 2000
CONTROL RIST
Control
(NaCl 0.97% IP
(20 days)
L-NAME (1 mg/kg IPV)
RIST POST-L-NAME
SIN-1 (5 mg/kg IPV)
RIST POST-SIN-1
Hepatic GSH
Quantification [6]

Figure 2(a)



Control Group (n=6): L-NAME (1mg/kg, ipv) reduces the RIST Index from 260.2 ± 15.6 mg glucose /kg to 121.2 ± 12.8 mg glucose /kg (52.3±5.8% inhibition). SIN-1(5mg/kg, ipv) restores insulin response with a RIST index of 258.1 ±18.5 mg glucose /kg. *** = $p<0.001$

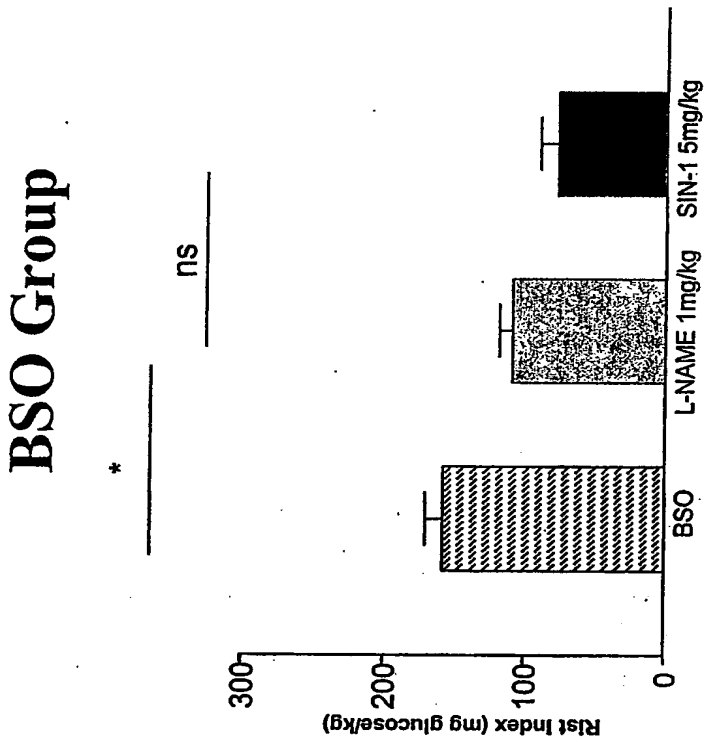


Figure 2(b)

BSO Group (n=5): The control RIST index was 158.4± 12.2 mg glucose /kg. Intraportal administration of L-NAME(1mg/kg) reduced significantly the RIST Index to 109.8± 9.1mg glucose /kg.Ipv administration of SIN-1 did not reverse the RIST Index to control values. *= p<0.05; ns= non significant

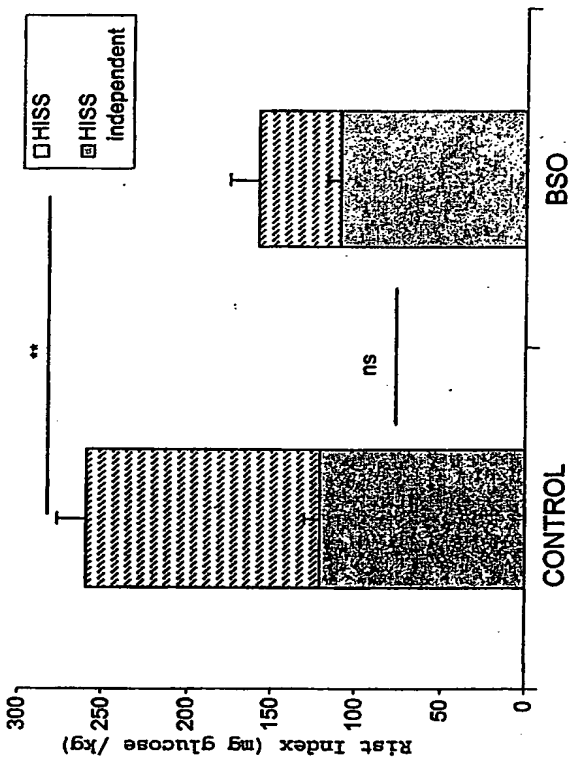


Figure 2(c)

HISS-dependent and HISS-independent components of insulin action in BSO and control groups. HISS-independent components are not different in both groups. HISS is significantly reduced in BSO group (49.3 ±8.56 mg glucose /kg) compared to control group (138.9 ± 22.8 mg glucose /kg) corresponding to a decrease of 64.4% of HISS action. **=p<0.01; ns= non significant

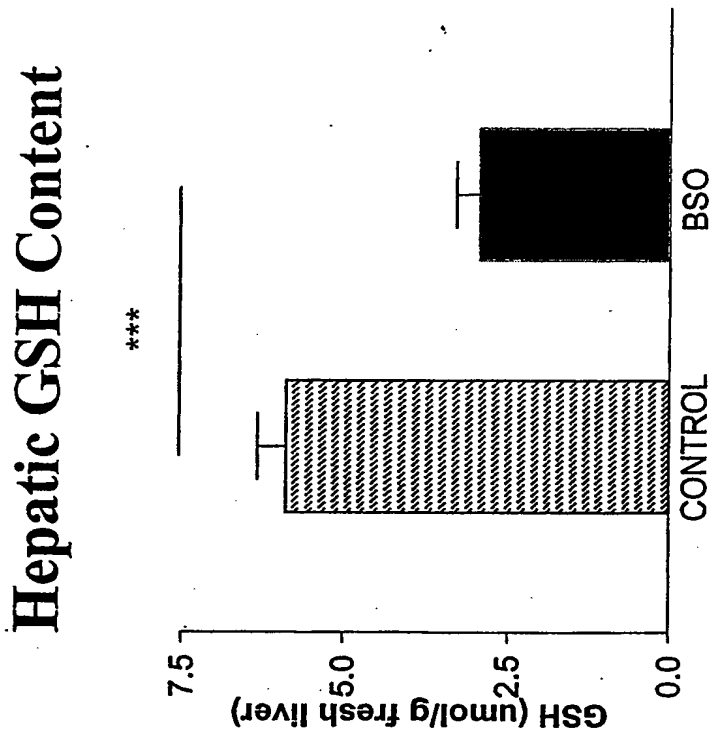


Figure 2(d)

Hepatic glutathione content in BSO (n=5) and control (n=6) groups. In control group hepatic GSH content was significantly higher (5.66 ± 0.1umol/g fresh liver) than in BSO group (2.96 ± 0.4umol/g fresh liver). Hepatic GSH content was decreased by 48.3 ± 6.9% in BSO group.
***=p<0.001